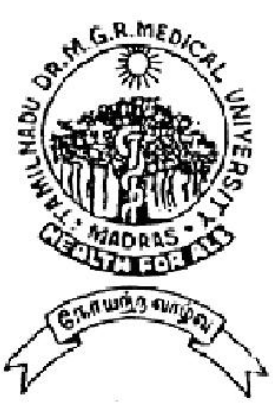
A STUDY ON THE PROFILE OF VARIOUS SCORING SYSTEMS IN ASSESSING THE SEVERITY OF ACUTE PANCREATITIS

DISSERTATION SUBMITTED FOR

M.D GENERAL MEDICINE

BRANCH – I

APRIL 2012



THE TAMILNADU

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CERTIFICATE

This is to certify that the dissertation entitled **"A STUDY ON THE PROFILE OF VARIOUS SCORING SYSTEMS IN ASSESSING THE SEVERITY OF ACUTE PANCREATITIS "** is the bonafide work of **Dr. NISHAD RAVEENDRAN,** in partial fulfillment of the university regulations of the Tamil Nadu Dr. M.G.R. Medical University, Chennai, for M.D General Medicine Branch I examination to be held in April 2012.

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DECLARATION

I, **Dr. NISHAD RAVEENDRAN,** solemnly declare that, I carried out this dissertation **"A STUDY ON THE PROFILE OF VARIOUS SCORING SYSTEMS IN ASSESSING THE SEVERITY OF ACUTE PANCREATITIS"** is a bonafide record of work done by me at the Department of General Medicine, Govt. Rajaji Hospital, Madurai, under the guidance of **Dr.G.Bagialakshmi M.D** Associate Professor, Department of General Medicine, Madurai Medical college, Madurai.

This dissertation is submitted to The Tamil Nadu Dr. M. G. R. Medical University, Chennai in partial fulfillment of the rules and regulations for the award of M.D Degree General Medicine Branch-I examination to be held in April 2012.

Place: Madurai

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ACKNOWLEDGEMENT

I would like to thank **DEAN**, Madurai Medical College, for permitting me to utilise the hospital facilities for the dissertation.

I also extend my sincere thanks to **Prof.Dr.MOSES .K.DANIEL M.D,**Head of the Department and Professor of Medicine for his constant support during the study.

I would like to express my deep sense of gratitude and thanks to my Unit Chief and Associate Professor Medicine, **Dr.G.BAGIALAKSHMI M.D.,** for her valuable suggestions and excellent guidance during the study.

I express my sincere thanks to **Dr.L.THAYUMANAVAN, M.D., D.M,** Professor of Medical Gastroenterology for permitting me to utilise thefacilities in the Department for the purpose of this study and guiding me with enthusiasm throughout the study period.

I thank the Assistant Professors of my Unit **Dr.S.PEER MOHAMMEDM.D,** and **Dr.K.PREMKUMAR, M.D.,** and Assistant Professors of Medical Gastroenterology **Dr.R.RAMANI M.D,D.M** and **Dr.M.KANNAN M.D,D.M** for their valid comments and suggestions.

Finally, I thank the patients for their extreme patience and co-operation

CONTENTS

ABSTRACT

Background: Acute pancreatitis is one of the most common cause of abdominal pain. About 10to 20% of patients will progress to Severe Acute Pancreatitis (SAP) with a mortality rate of 6 to 10 %.Individual patient's response to pancreatitis is highly variable. Because of this, it is of utmost importance to predict who is at the risk of developing severe pancreatitis as this will help to institute more intensive treatment. This will in turn improve the mortality. For this purpose various scoring systems are used. This study compares the strength of various prognostic factors in assessing the severity of acute pancreatitis. We also compared the sensitivity and specificity of various scoring systems in assessing the severity and mortality of acute pancreatitis.

Methods: A prospective, observational, clinical and investigational study was carried out in 50 patients admitted with acute pancreatitis in Government Rajaji Hospital Madurai. Twelve individual parameters -Age, WBC count, RBS, Blood Urea, S.Creatinine, PaO2, S.Calcium, S.LDH, S.bilirubin, S.Albumin,S.AST and S.ALT were assessed for their strength of association with severity of acute pancreatitis. Five scoring systems APACHE II, Ranson's, Imrie's, Bank's, Pitchumani & Agarwal system were compared for their sensitivity and specificity for assessing the severity and mortality of acute pancreatitis.

Results: Among the 50 patients 18 patients developed Severe Acute Pancreatitis (SAP) marked by evidence of end organ failure, local complications like psuedocyst, and /or prolonged ICU stay of more than 7 days. Among this 8 patients died. Among the individual parameters B.Urea,S.Creatinine,PaO2 and WBC count, Age,S.Calcium and S.LDH levels showed a significant association with severity of acute pancreatitis. APACHE II systems had a sensitivity of 77.78% and specificity of 96.88% in predicting severity.Sensitivity and specificity of Ranson's score was

83.33% and 96.88% respectively. Imrie’s score had a sensitivity of 55% and specificity of 100% .Both Bank’s score and Pitchumani score had comparatively low sensitivity and specificity.

Conclusion:APACHE II score and Ranson’s score are the best scoring systems in predicting the severity of acute pancreatitis.Among individual parameters Hypoxia, Acute renal failure, leukocytosis, advancing age ,hypocalcemia and increase in serum LDH levels were the factors significantly associated with Severe Acute Pancreatitis(SAP).

INTRODUCTION

Acute pancreatitis is one of the most important cause of abdominal pain. Its incidence varies from 5-80 per 100000 population[59]. The clinical course of acute pancreatitis is usually mild and often resolves without sequele. Between 10-20%[14] patients experiences severe acute pancreatitis (SAP) attacks resulting in intense inflammatory response, a variety of local and systemic complications which can lead to prolonged hospital stay with significant morbidity and mortality. The mortality ranges between 6-10%[64].

Individual patient's response to pancreatitis is often variable and highly unpredictable. But early recognition of a patient who is more likely to progress to Severe Acute Pancreatitis(SAP) is important because these patients may need more aggressive treatment including surgical interventions. This will in turn translate into improved outcome.

This has led to the development of various biochemical markers and scoring system for predicting the severity of acute pancreatitis. Several scoring systems has developed for this which includes Ranson's, Imrie's, Bank's, Pitchumani and Agarwal etc.

Eventhough it was originally designed to predict the intensive care unit survival, Apache II system is also used for this purpose. An ideal predictive criteria should be simple, non invasive and quantitative and the assessment tests should be readily available at the time of diagnosis.

In this study we are analyzing the strength of various biochemical parameters in predicting the severity of acute pancreatitis and also comparing the accuracy of various scoring system in predicting the mortality and severity of acute pancreatitis.

AIM OF THE STUDY

1. To compare the various scoring systems with Apache II in predicting the severity of acute pancreatitis.
2. To analyze whether any single parameter as an Index of severity of acute pancreatitis.
3. To compare our study with published literature world wide.

REVIEW OF LITERATURE

Acute pancreatitis has been recognized since time immemorial and has been described as the most terrible of all calamities that occur in connection with the abdominal viscera[36]. In 1889, Reginald Fitz gave the classic clinical and pathological description of acute pancreatitis[37].

The pancreas is a gland located in the upper, posterior abdomen and is responsible for insulin production (endocrine pancreas) and the manufacture and secretion of digestive enzymes (exocrine pancreas) leading to carbohydrate, fat, and protein metabolism. Approximately 80% of the gross weight of the pancreas supports exocrine function, while the remaining 20% is involved with endocrine function.

As mentioned, the principal function of the exocrine pancreas is to make food-digesting enzymes. Enzymes are produced within the pancreatic acinar cells, packaged into storage vesicles called zymogens, and then released via the pancreatic ductal cells into the

pancreatic duct, where they are secreted into the small intestine to begin the metabolic process.

The incidence of pancreatitis varies in different countries and depends on cause, e.g., alcohol, gallstones, metabolic factors, and drugs. The estimated incidence in the United States it is 40/100000 population per year[22]. Worldwide, the incidence of acute pancreatitis ranges between 5 and 80 per 100,000 population, with the highest incidence recorded in the United States and Finland[59].

Race

The hospitalization rates of patients with acute pancreatitis per 100,000 population are 3 times higher for blacks than whites. These racial differences are more pronounced for males than females.

Sex

In general, acute pancreatitis affects males more often than females.

The etiology in males is more often related to alcohol; in females, to biliary tract disease.

Idiopathic pancreatitis has no clear predilection for either sex. Pancreatic inflammatory disease may be classified as[1]

(1) Acute pancreatitis.

(2) Chronic pancreatitis.

The pathologic classification[1]

1. **Interstitial pancreatitis**, also called edematous pancreatitis which is usually a mild and self-limited disorder.

2. **Necrotizing pancreatitis**, occurs in about 20%–30% of all patients with acute pancreatitis in which the degree of pancreatic necrosis correlates with the severity of the attack and its systemic manifestations. It is characterized by a protracted clinical course, a high incidence of local complications, and a high mortality rate. Parenchymal pancreatic injury is the pathologic hallmark of this form of the disease.

Etiology and Pathogenesis

There are many causes of acute pancreatitis but the mechanisms by which these conditions trigger pancreatic inflammation have not been identified.

Gallstones continue to be the leading cause of acute pancreatitis in most series (30–60%).

Alcohol is the second most common cause, responsible for 15–30% of cases in the United States.

Hypertriglyceridemia is the cause of acute pancreatitis in 1.3–3.8% of cases; serum triglyceride levels are usually >11.3 mmol/L (>1000 mg/dL).

Acute pancreatitis occurs in 5–20% of patients following **endoscopic retrograde cholangiopancreatography (ERCP).**

Approximately 2–5% of cases of acute pancreatitis are drug-related. Drugs cause pancreatitis either by a hypersensitivity reaction or by the generation of a toxic metabolite, although in some cases it is not clear which of these mechanisms is operative.

Drugs definitely associated with acute pancreatitis include azathioprine, sulfonamides, sulindac, tetracycline, valproic acid, didanosine, methyldopa, estrogens, furosemide, 6-mercaptopurine, pentamidine, 5-aminosalicylic acid compounds, corticosteroids, and octreotide.

Infection (<1%):Viral causes include mumps, Epstein-Barr,HIV, coxsackievirus, echovirus, varicella-zoster, and measles. Bacterial causes include Mycoplasma pneumoniae, Salmonella, Campylobacter, and Mycobacterium tuberculosis.

Other Cause:

Hereditary pancreatitis (< 1%)

Hypercalcemia (< 1%)

Developmental abnormalities of the pancreas (< 1%)

Hypertriglyceridemia (< 1%)

Toxins (<1%):Exposure to organophosphate insecticide can cause acute pancreatitis.

Tumor (< 1%):Obstruction of the pancreatic ductal system by a pancreatic ductal carcinoma, ampullary carcinoma, islet cell tumor, solid pseudotumor of the pancreas, sarcoma, lymphoma, cholangiocarcinoma, or metastatic tumor can cause acute pancreatitis.

Postoperative (< 1%)

Vascular abnormalities (< 1%)

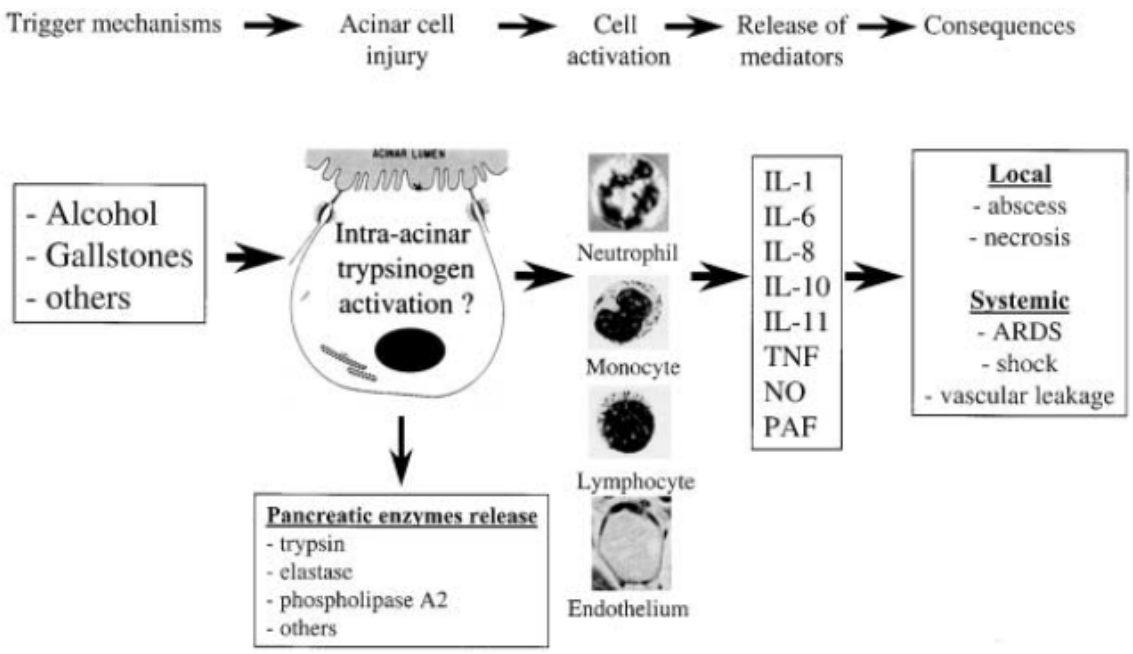
Autoimmune pancreatitis (< 1%)

In up to 10% of cases, the cause of pancreatitis remains unknown (idiopathic).

Pancreatic Injury: Pathophysiology

It has been assumed that the initial triggering event occurs at the cellular level and is based on premature activation of pancreatic enzymes leading to autodigestion of the pancreatic parenchyma and peripancreatic tissues. The mechanism by which pancreatic enzymes

are activated outside the intestinal tract remains obscure. Intraparenchymal and extrapancreatic extravasation of these activated digestive enzymes is responsible for tissue injury and for damage to the pancreatic vascular network.



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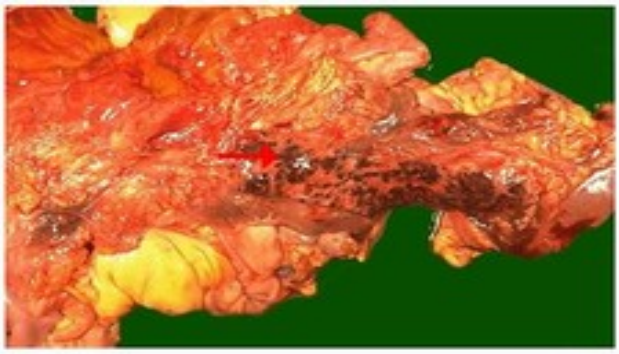
Pathologic examination of severe pancreatitis has shown extensive interstitial fat necrosis, necrotizing vasculitis with occlusions and thrombosis of small feeding arteries and draining veins, areas of hemorrhage, and devitalized pancreatic parenchyma.

Similar findings are present in variable degrees in extrapancreatic retroperitoneal fatty tissue.

Necrosis occurs early, within the first 24–48 hours, and it can be diffuse or patchy or superficial or deep, and it may affect any part of the pancreatic gland.



This is an example of acute pancreatitis. The pancreas is swollen and does not show the typical tan, lobulated architecture. Instead, it has areas of hemorrhagic necrosis that appear as blotchy black red areas at the mid right of the photograph.

CLINICAL FEATURES

Abdominal pain is the major symptom of acute pancreatitis. Pain may vary from a mild and tolerable discomfort to severe, constant, and incapacitating distress. Characteristically, the pain, which is steady and boring in character, is located in the epigastrium and periumbilical region and often radiates to the back as well as to the chest, flanks, and lower abdomen. The pain is frequently more intense when the patient is supine, and patients often obtain relief by sitting with the trunk flexed and knees drawn up.

Nausea, vomiting, and abdominal distention due to gastric and intestinal hypomotility and chemical peritonitis are also frequent complaints.

Physical examination

Low-grade fever,

Tachycardia,

Hypotension,

Jaundice occurs infrequently; when present, it usually is due to edema of the head of the pancreas with compression of the intrapancreatic portion of the common bile duct.

Erythematous skin nodules due to subcutaneous fat necrosis may occur.

In 10–20% of patients, there are pulmonary findings, including basilar rales, atelectasis, and pleural effusion, the latter most frequently left-sided.

Abdominal tenderness and muscle rigidity are present to a variable degree, but, compared with the intense pain, these signs may be unimpressive.

Bowel sounds are usually diminished or absent. An enlarged pancreas with organized necrosis or a pseudocyst may be palpable in the upper abdomen.

A faint blue discoloration around the umbilicus (Cullen's sign) may occur as the result of hemoperitoneum, and a blue-red-purple or green-brown discoloration of the flanks (Turner's sign) reflects tissue catabolism of hemoglobin. The latter two findings, which are uncommon, indicate the presence of a severe necrotizing pancreatitis.

Laboratory Data

The diagnosis of acute pancreatitis is usually established by the detection of an increased level of serum amylase. Values

threefold or more above normal virtually clinch the diagnosis if overt salivary gland disease and gut perforation or infarction are excluded. However, there appears to be no definite correlation between the severity of pancreatitis and the degree of serum amylase elevation. After 48–72 h, even with continuing evidence of pancreatitis, total serum amylase values tend to return to normal. However, pancreatic isoamylase and lipase levels may remain elevated for 7–14 days

Serum lipase activity increases in parallel with amylase activity. Measurement of both enzymes is important as serum amylase tends to be higher in gallstone pancreatitis and serum lipase higher in alcohol-associated pancreatitis. A threefold elevated serum lipase value is usually diagnostic of acute pancreatitis; these tests are especially helpful in patients with nonpancreatic causes of hyperamylasemia.

Markedly increased levels of peritoneal or pleural fluid amylase [>1500 nmol/L (>5000 U/dL)] are also helpful, if present, in establishing the diagnosis.

Leukocytosis (15,000–20,000 leukocytes per L) occurs frequently. Patients with more severe disease may show

hemoconcentration with hematocrit values >44% because of loss of plasma into the retroperitoneal space and peritoneal cavity. Hemoconcentration may be the harbinger of more severe disease, i.e., pancreatic necrosis.

Hyperglycemia is common and is due to multiple factors, including decreased insulin release, increased glucagon release, and an increased output of adrenal glucocorticoids and catecholamines.

Hypocalcemia occurs in ~25% of patients, and its pathogenesis is incompletely understood.

Hyperbilirubinemia [serum bilirubin > 68 mol/L (>4.0 mg/dL)] occurs in ~10% of patients. However, jaundice is transient, and serum bilirubin levels return to normal in 4–7 days.

Serum alkaline phosphatase and aspartate aminotransferase (AST) levels are also transiently elevated and parallel serum bilirubin values.

Markedly elevated serum lactic dehydrogenase (LDH) levels [>8.5 mol/L (>500 U/dL)] suggest a poor prognosis.

Serum albumin is decreased to 30 g/L (3.0 g/dL) in ~10% of patients; this finding is associated with more severe pancreatitis and a higher mortality rate.

Hypertriglyceridemia occurs in 15 to 20% of patients, and serum amylase and lipase levels in these individuals are often spuriously normal.

Approximately 25% of patients have hypoxemia (arterial P_{O2} <60 mmHg), which may herald the onset of ARDS.

Abdominal ultrasonography

This is the most useful initial test in determining the etiology of pancreatitis and is the technique of choice for detecting gallstones.

Abdominal CT scanning

This is generally not indicated for patients with mild pancreatitis unless a pancreatic tumor is suspected (usually in elderly patients).

CT scanning is always indicated in patients with severe acute pancreatitis and is the imaging study of choice for assessing complications.

Abdominal CT scans also provide prognostic information based on the following grading scale developed by Balthazar[17]:

A - Normal

B - Enlargement

C - Peripancreatic inflammation

D - Single fluid collection

E - Multiple fluid

The chances of infection and death are virtually nil in grades A and B but steadily increase in grades C through E. Patients with grade E pancreatitis have a 50% chance of developing an infection and a 15% chance of dying.

Magnetic resonance cholangiopancreatography

Heavily T-2–weighted images provide a noninvasive image of the biliary and pancreatic ducts. Although not as sensitive as ERCP, MRCP is safer, noninvasive, and fast, and it provides images useful in guiding clinical care decisions. This modality should be used if choledocholithiasis is suspected.

Diagnosis

Any severe acute pain in the abdomen or back should suggest acute pancreatitis. The diagnosis is usually entertained when a patient with a possible predisposition to pancreatitis presents with severe and constant abdominal pain, nausea, emesis, fever, tachycardia, and abnormal findings on abdominal examination. Laboratory studies frequently reveal leukocytosis, hypocalcemia, and hyperglycemia. The diagnosis is usually confirmed by the

finding of a threefold or greater elevated level of serum amylase and/or lipase.

Acute Pancreatitis: Treatment

In most patients (85–90%) with acute pancreatitis, the disease is self-limited and subsides spontaneously, usually within 3–7 days after treatment is instituted.

Conventional measures include (1) analgesics for pain, (2) IV fluids and colloids to maintain normal intravascular volume, and (3) no oral alimentation.

Nasogastric suction offers no clear-cut advantages in the treatment of mild to moderately severe acute pancreatitis.It has been demonstrated that CCK-stimulated pancreatic secretion is almost abolished in four different experimental models of acute pancreatitis. This finding probably explains why drugs to block pancreatic secretion in acute pancreatitis have failed to have any therapeutic benefit. For this and other reasons, anticholinergic drugs are not indicated in acute pancreatitis.

Total parentral nutrition / artificial nutrition

Where it is feasible and when enteral nutrition is contraindicated, use of amino acid infusion about 1-1.5 g/kg/day and

the use of intravenous lipids, unless contraindicated by elevated triglyceride levels, in several cases especially in sepsis cases has helped to decrease morbidity and mortality. Recent studies on intensive care patients with trauma and sepsis showed that enteral feeding was associated with a reduction in the acute phase response and the severity of septic complications compared to total parentral nutrition . A clear liquid diet is frequently started on the third to sixth day and a regular diet by the fifth to seventh day. The decision to reintroduce oral intake is usually based on the following criteria: (1) a decrease in or resolution of abdominal pain; (2) the patient is hungry; and (3) organ dysfunction, if present, has resolved. Elevation of serum amylase/lipase or persistent inflammatory changes seen on CT scans should not discourage feeding a hungry asymptomatic patient.

Role of Antibiotics

The benefit of antibiotic prophylaxis in the treatment of necrotizing acute pancreatitis remains controversial. Although the optimal drugs and duration of therapy remain incompletely defined, the current recommendation in patients with necrotizing acute

pancreatitis is the use of a systemic antibiotic such as Imipenem-Cilastin, 500 mg thrice daily for 7 days.

Several other drugs have been evaluated by prospective controlled trials and found ineffective in the treatment of acute pancreatitis. These drugs includes glucagon, H_2 blockers, protease inhibitors such as aprotinin, glucocorticoids, calcitonin, nonsteroidal anti-inflammatory drugs (NSAIDs), and lexipafant, a platelet-activating factor inhibitor.

A recent meta-analysis of somatostatin, octreotide, and the antiprotease gabexate mesylate in therapy of acute pancreatitis suggested (1) a reduced mortality rate but no change in complications with octreotide, and (2) no effect on the mortality rate but reduced pancreatic damage with gabexate.

SURGICAL TREATMENT

Most patients with acute pancreatitis do not require surgical treatment of the pancreatic disease although many will subsequently undergo cholecystectomy.

Indications for intervention in pancreatic necrosis[8]

The decision to intervene depends on the clinical picture (evidence of sepsis) and demonstration by CT of pancreatic or

peripancreatic necrosis. There is agreement that all patients with infected necrosis require intervention by radiological or surgical drainage.The infection may be diagnosed either by the presence of gas within the pancreatic collection or by fine needle aspiration. Patients with infected necrosis will require intervention to completely debride all cavities containing necrotic material.

Radiological drainage[8]

In one report, 31 patients with pancreatic abscess were managed by percutaneous drainage. There was a 31% primary success rate. Freeny et al also reported encouraging results: in 34 patients nearly half had successful treatment by catheter drainage and only nine required surgical drainage. This suggests that percutaneous wide bore drainage may be sufficient for the treatment of infected necrosis.

COMPLICATIONS[1]

Local Complications

Necrosis

1. Sterile
2. Infected
3. Organized

Pancreatic fluid collections

Pancreatic abscess

Pancreatic pseudocyst : This is a collection of pancreatic fluid enclosed by a wall of granulation tissue and requires 4 or more weeks to develop.Complications of Psuedocyst includes:

1. Pain
2.Rupture
3.Hemorrhage
4. Infection
5. Obstruction of gastrointestinal tract (stomach, duodenum, colon)

Pancreatic ascites : which could be due to

1.Disruption of main pancreatic duct
2.Leaking pseudocyst

Involvement of contiguous organs by necrotizing pancreatitis

1. Massive intraperitoneal hemorrhage
2.Thrombosis of blood vessels (splenic vein, portal vein)
3. Bowel infarction
4.Obstructive jaundice

Intra-abdominal infections

Within the first 1-3 weeks, fluid collections or pancreatic necrosis can become infected and jeopardize clinical outcome.

From 3-6 weeks, pseudocysts may become infected or a pancreatic abscess may develop.

A pancreatic abscess is a circumscribed intra-abdominal collection of pus, within or in proximity to the pancreas. It is believed to arise from localized necrosis, with subsequent liquefaction that becomes infected.

Pancreatic necrosis

This is a nonviable area of pancreatic parenchyma that is often associated with peripancreatic fat necrosis and is principally diagnosed with the aid of dynamic spiral CT scans. Sterile pancreatic necrosis is usually treated with aggressive medical management, whereas almost all patients with infected pancreatic necrosis require surgical debridement or percutaneous drainage if they are to survive.

Systemic Complications

Pulmonary:

:Pleural effusion,Atelectasis, Pneumonitis,

:Adult respiratory distress syndrome,

:Mediastinal abscess.

Cardiovascular:

:Hypotension, Hypovolemia, Sudden death,

: Pericardial effusion,

:Nonspecific ST-T changes in electrocardiogram

simulating myocardial infarction .

Hematologic :

:Disseminated intravascular coagulation.

Gastrointestinal :

:Peptic ulcer disease,Erosive gastritis,

:Hemorrhagic pancreatic necrosis with erosion in to major blood vessels,

:Portal vein thrombosis, variceal hemorrhage

Renal :

: Oliguria,Azotemia, Acute tubular necrosis,

:Renal artery and/or renal vein thrombosis

Metabolic:

:Hyperglycemia,Hypertriglyceridemia, Hypocalcemia,

:Encephalopathy,

: Sudden blindness (Purtscher's retinopathy)

Central nervous system:

: Psychosis, Fat emboli

Fat necrosis: Subcutaneous tissues (erythematous nodules)

Mortality/Morbidity

The overall mortality rate of patients with acute pancreatitis is 10-15%. This rate has been falling over the last two decades as improvements in supportive care have been initiated.

In patients with severe disease (organ failure), the mortality rate is approximately 30%. This rate in mortality has not dropped in the last 10 years.

In the first week of illness, most deaths result from multiorgan system failure. In subsequent weeks, infection plays a more significant role, but organ failure still constitutes a major cause of mortality.

Multiple Prognostic Indices

Once the diagnosis of acute pancreatitis is established, the treatment of patients depends on the early assessment of disease severity. This assessment, based on objective parameters, is crucial for predicting clinical complications and for identifying potentially lethal attacks.

For many years, it has been recognized that obvious alterations of clinical parameters and some abnormal results of routine laboratory tests are often present in patients with severe pancreatitis. For instance, a low serum calcium level (7.5 mg/dL [1.88 mmol/L])

detected in the background in cases of an acute attack of pancreatitis is a worrisome sign that is seen mainly in patients with severe disease [17].Furthermore, it has been shown that the risk of death is increased in patients in whom the serum glucose level is above 250 mg/dL (13.9 mmol/L) and the serum creatinine level after rehydration is above 2 mg/dL (177 mmol/L)[17]. Signs of multiorgan failure and some specific abnormal clinical and laboratory findings can help identify patients with a severe, potentially lethal form of disease. The presence of one or several signs of distal organ failure was associated with a 50% mortality rate in the series of Bank et al. None of the individual clinical or laboratory parameters, while useful in clinical practice, are sufficiently sensitive or specific to help identify most patients with necrotizing pancreatitis.

In the attempt to overcome these deficiencies, various scoring systems that combine clinical and laboratory parameters have been devised to help identify patients with severe pancreatitis. These scoring systems use the number of specific abnormalities, called prognostic signs, grave signs, risk factors, or objective indicators, to stage acute pancreatitis. It should be emphasized that these physiologic alterations reflect systemic abnormalities; they do not

correlate well with severity and extent of local disease, and they certainly do not have diagnostic specificity, because they can be seen in a variety of other conditions.

The first numeric system, proposed by Ranson et al in 1974[29] (Ranson system), is still the most widely used. Originally, the Ranson score was created from a retrospective review of one institution's experience with pancreatitis. The authors examined 43 variables from 100 consecutive patients with pancreatitis and found 11 different variables that correlated with subsequent mortality and morbidity.

It is based on 11 objective signs: five determined initially, and six within 48 hours. With an increased number of risk factors, there is a corresponding increase in the morbidity and mortality rates. In patients with fewer than three positive signs, there is no mortality, three or more than three positive signs have increased mortality, while in patients with six or more signs the mortality rate is over 50%. Individuals with more than six grave signs usually have necrotizing pancreatitis.

At admission:

1. Age in years > 55 years
2. White blood cell count > 16000 cells/mm^3
3. Blood glucose > 10 mmol/L (> 200 mg/dL)
4. Serum AST > 250 IU/L
5. Serum LDH > 350 IU/L

At 48 hours:

1. Calcium (serum calcium < 2.0 mmol/L (< 8.0 mg/dL)
2. Hematocrit fall > 10%
3. Oxygen (hypoxemia P_{O2} < 60 mmHg)
4. BUN increased by 1.8 or more mmol/L (5 or more mg/dL) after IV fluid hydration.
5. Base deficit (negative base excess) > 4 mEq/L
6. Sequestration of fluids > 6 L

Although the Ranson score has been widely purported to be a valid measure of outcome, it was never validated prospectively by its creators or tested in any type of large multicenter trial subsequent to its inception. Others disadvantage is that even if the Ranson score were an accurate predictor, a 48-hour period is required before the total score can be tabulated.

Alternative grading systems, each using different parameters, have since been constructed, with a prognostic capability generally similar to that of the Ranson system. The Glasgow original or modified system, the Simplified Acute Physiology or SAP, score, and simplified prognostic criteria have been used.

Blamey et al introduced a modification of the Ranson system[19], based on eight prognostic criteria. They omitted hematocrit level, base deficit, age, and fluid sequestration but included serum albumin level of less than 32 g/L as an important criterion of severity. Despite modifications and fine tuning, however, the overall sensitivity of the aforementioned numeric systems in the initial staging of an attack of pancreatitis ranges from 57% to 85%, with a specificity of 68%–85% .

Modified Glasgow system by Imrie :

A score ≥3 suggestive of SAP

During Initial 48 hours

WBC count >15 x 10^9/L (15 x 10^3/microlitre)

Serum albumin <32 g/L (3.2 g/dL)

Arterial PO2 on room air <8 kPa (60 mmHg)

Serum calcium <2 mmols/L (8 mg/dL)

Blood glucose >10.0 mmols/L (180 mg/dL)

Serum LDH >600 units/L

Serum urea nitrogen >16.1 mmols/L (45 mg/dL)

ALT/AST>200 U/L

Severe Acute Pancreatitis (SAP) as Defined by Atlanta Symposium

The International Symposium, held in Atlanta, in 1992, established a clinically based classification system for acute pancreatitis. According to the Atlanta Symposium, acute pancreatitis was defined as an acute inflammatory process of the pancreas that may also involve peripancreatic tissues and/or remote organ systems. Criteria for severity included organ failure (particularly shock, pulmonary insufficiency, and renal failure) and/or local complications (especially pancreatic necrosis but also including abscess and pseudocyst). Early predictors of severity within 48 h of initial hospitalization included Ranson signs and APACHE-II points

Early Prognostic Signs

Ranson signs ≥3

APACHE-II score ≥8

Organ Failure marked by

1. Shock–systolic pressure <90 mmHg
2. PaO2 ≤60 mmHg
3. Creatinine >2.0 mg/L after rehydration

4. Gastrointestinal bleeding >500 cc/24 h

And/Or

Local Complications

1. Necrosis
2. Abscess
3. Pseudocyst

More recently, the Acute Physiology and Chronic Health Evaluation (APACHE II) assessment and monitoring system has become popular, because it is considered to be more reliable[41].The acute physiology score and the chronic health evaluation (APACHE) were used in the first major attempts to quantify the severity of the illness in ICU patients, by Knaus et al in 1981 and this was later modified in 1985 by the same author as APACHE II[39,40]

It contains 12 continuous variables from the original APACHE system and also takes into account the age of the patient, the pre-morbid conditions and the Glasgow coma scale (GCS). The major advantage of the APACHE II scoring system, as compared to the other systems, is that it can be used in monitoring the patient's response to therapy while the Ranson and the Glasgow scales are mainly meant for the assessment at presentation .

The APACHE II scoring system takes into account 12 variables which include, (1) Body temperature, (2) mean arterial pressure (mm Hg), (3) Heart rate(HR), (4) respiratory rate (R.R/mt), (5) Oxygenation (mm Hg), (6) PH, (7) Na+ (mmol/l), (8) K+ (mmol/l), (9) Creatinine (mg/dl), (10) Haematocrit, (11) total leucocyte count and the (12) Glasgow coma score.

To eliminate the problem of the missing values and concerns about the assumption that an unmeasured variable was normal, the measurement of all the 12 variables was made mandatory for the usage of APACHE II. The recorded values of the variables are based on the most derange values during the past 24 hours .Because age and severe chronic health problems reflects diminished physiological reserve, they have been directly incorporated into

APACHE II

	+ 4	+3	+2	+1	0	+1	+2	+3	+4
1 Rectal temp (◦C)	*>41*	39–40.9		38–38.9	36–38.4	34–35.9	32–33.9	30–31.9	*<29.9*
2 Mean arterial pressure (mmHg)	*>160*	130–159	110–129		70–109		50–69		*<49*
3 Heart rate (bpm)	*>180*	140–179	110–139		70–109		55–69	40–54	*<39*
4 Respiratory rate (bpm)	*>50*	35–49		25–34	12–24	10–11	6–9		*<5*
5 Oxygen delivery (mL/min)	*>500*	350–499	200–349		*<200*				
6 PO2 (mmHg)					*>70*	61–70		55–60	*<55*
7 Arterial pH	*>7.7*	7.6–7.69		7.5–7.59	7.3–7.49		7.25–7.3	7.15–7.2	*<7.15*
8 Serum sodium (mmol/L)	*>180*	160–179	155–159	150–154	130–149		120–129	111–119	*<110*
9 Serum potassium (mmol/L)	*>7*	6–6.9		5.5–5.9	3.5–5.4	3–3.4	2.5–2.9		*<2.5*
10 Serum creatinine (mg/dL)	*>3.5*	2–3.4	1.5–1.9		0.6–1.4		*<0.6*		
11 Hematocrit (%)	*>60*		50–59.9	46–49.9	30–45.9		20–29.9		*<20*
12 White cell count (103/mL)	*>40*		20–39.9	15–19.9	3–14.9		1–2.9		*<1*

			Age Points						
Age									Points
<44									0
45–54									2
55–64									3
65–74									5
>75									6

	Chronic Health Points	
History of Severe Organ Insufficiency		Points
Nonoperative patients		5
Emergency postoperative patients		5
Elective postoperative patients		2

The system is complex and more difficult to perform, because 12 physiologic measurements are used. The higher the total score, the more severe the pancreatitis, with a corresponding increase in morbidity and mortality. It has been suggested that a cutoff APACHE II score of greater than 8 indicates severe pancreatitis. The major advantage of the APACHE II numeric system, as compared with the other systems, is that it can be used throughout the patient's hospital course in monitoring the patient's response to therapy .The accuracy of the APACHE II system at admission for the assessment of the severity of pancreatitis has been about 75%. The test is useful as an early prognostic indicator of disease severity to help identify patients for intensive care unit treatment. After 48 hours, APACHE II scores are comparable with Ranson system scores in distinguishing mild from severe pancreatitis, with an accuracy of about 70%–80% .

CT Severity Index[17]

The CT severity index is an attempt to improve the early prognostic value of CT in cases of acute pancreatitis. Patients with grade A–E pancreatitis are assigned zero to four points plus two points for necrosis of up to 30%, four points for necrosis of 30%–

50%, and six points for necrosis of more than 50%. For instance, a patient with CT grade D is assigned three points; if, in addition, the patient has more than 50% necrosis, an additional six points are assigned, for a total index score of 9. There was a statistically significant correlation, with a continuous increasing incidence of morbidity and mortality in patients stratified according to CT severity index groups. Patients who had a severity index of 0 or 1 exhibited a 0% mortality rate and no morbidity, while patients with severity index of 2 had no mortality and a 4% morbidity rate. In contrast, a severity index of 7–10 yielded a 17% mortality rate and a 92% complication rate .

One of the other most commonly used scoring system is **Banks System**[27]

A score of ≥ 1 suggestive of SAP

Cardiac	-	Shock / tachycardia > 130, arrhythmia
Pulmonary	-	Dyspnoea, PaO2 < 60 mm, ARDS
Renal	-	Urine output < 50 ml / hr, Rising blood urea / Creatinine
Metabolic	-	Low or falling calcium, pH, albumin decrease

Hematological - Falling PCV, DIC

Neurological - Irritability, confusion, localizing signs

Hemorrhagic - On signs or peritoneal tap

Tense distention - Severe ileus, fluid ++

Pitchumani and Agarwal[28]

A score ≥1 suggestive of SAP

During initial 48 hrs

Cardiac - BP < 90 mmHg / tachycardia > 130 BPM

Pulmonary - PO2 < 60 mm Hg

Renal - Urine output < 50 ml / min

Metabolic - Calcium < 8 mg / dl or and albumin < 3.2 g/dl

The newer biomarkers in predicting the severity includes various cytokines like IL-1,IL-6,IL-8,IL-10 and TNF-alpha.Various pancreatic products like lipase,procarboxypeptidases,pancreatitis associated peptide, trypsinogen 2 are also under study[74].

MATERIALS AND METHODS

Place of study : Dept. of General Medicine
Govt. Rajaji Hospital, attached to
Madurai Medical College, Madurai.

Type of study : Prospective, Observational, Clinical and Investigational study.

Ethical Committee: Ethical committee approval obtained.

Collaborating Department: Dept.of Medical Gastroenterology

Period of Study : From September 2010 to August 2011

Financial Support: Nil

Conflict of Interest: Nil

Selection and Details of study subjects :

In this study, 50 patients admitted to Medicine / Medical Gastroenterology / Surgery and Surgical Gastroenterology wards of Govt. Rajaji Hopsital, Madurai with acute pancreatitis was included randomly.

Inclusion Criteria :

The diagnosis of acute pancreatitis was based on

1. **Clinical criteria** : History of abdominal pain radiating to back and relieved by bending forward and associated with tenderness and guarding of upper abdomen.

2. **Radiographic evidence**

CT / USG findings suggestive of acute pancreatitis like pancreatic edema, pancreatic necrosis, peripancreatic fluid collection.

3. **Biochemical**

Serum Amylase greater than 3 times of normal

Exclusion criteria :

1. All those patients with chronic pancreatitis were excluded.
2. All the previously treated patients were excluded from our study.

METHODOLOGY

History taking and physical examinations was done in all patients. Physical examination included assessment of Glasgow coma scale, heart rate, blood pressure, mean arterial pressure, temperature, respiratory rate.

The following investigations were carried out

1. Packed cell volume (PCV)

2. Total WBC count (TC)
3. Platelet counts (PLC)
4. Random blood sugar (RBS)
5. Blood urea
6. Serum creatinine
7. Serum bilirubin
8. Alaline aminotransferase (ALT)
9. Aspartate Aminotransferase (AST)
10. Serum calcium (S.Ca++)
11. Serum Lactate Dehydrogenase (S.LDH)
12. Pa O2
13. Base Deficit: measured as 24 minus serum Bicarbonate level
14. Fluid Sequesteration

Based on these clinical and investigational parameters patients were assigned scores according to Apache II, Ranson's, Bank's Imries, Pitchumani and Agarwal scoring systems using data's from the first 48 hrs.

Ranson's Score: ≥3 was taken as predictor of severe pancreatitis

At admission:

1. Age in years > 55 years
2. White blood cell count > 16000 cells/mm^3
3. Blood glucose > 10 mmol/L (> 200 mg/dL)
4. Serum AST > 250 IU/L
5. Serum LDH > 350 IU/L

At 48 hours:

1. Calcium (serum calcium < 2.0 mmol/L (< 8.0 mg/dL)
2. Hematocrit fall > 10%
3. Oxygen (hypoxemia P_{O2} < 60 mmHg)
4. BUN increased by 1.8 or more mmol/L (5 or more mg/dL) after IV fluid hydration.
5. Base deficit (negative base excess) > 4 mEq/L
6. Sequestration of fluids > 6 L

Imries : A score ≥3 suggestive of SAP

During Initial 48 hours

1. WBC count >15 x 10^9/L (15 x 10^3/microlitre)
2. Serum albumin <32 g/L (3.2 g/dL)
3. Arterial PO2 on room air <8 kPa (60 mmHg)
4. Serum calcium <2 mmols/L (8 mg/dL)
5. Blood glucose >10.0 mmols/L (180 mg/dL)
6. Serum LDH >600 units/L
7. Serum urea nitrogen >16.1 mmols/L (45 mg/dL)
8. ALT/AST>200 U/L

Banks System[2] : A score of ≥ 1 suggestive of SAP

Cardiac - Shock / tachycardia > 130, arrhythmia

Pulmonary - Dyspnoea, PaO2 < 60 mm, ARDS

Renal - Urine output < 50 ml / hr, Rising blood urea / Creatinine

Metabolic - Low or falling calcium, pH, albumin decrease

Hematological- Falling PCV, DIC

Neurological- Irritability, confusion, localizing signs

Hemorrhagic- On signs or peritoneal tap

Tense distention- Severe ileus, fluid ++

Pitchumani and Agarwal[28] : A score ≥1 suggestive of SAP

During initial 48 hrs

Cardiac - BP < 90 mmHg / tachycardia > 130 BPM

Pulmonary - PO2 < 60 mm Hg

Renal - Urine output < 50 ml / min

Metabolic - Calcium < 8 mg / dl or and albumin < 3.2 g/dl

APACHE II Score > 8 suggestive of SAP

	+4	+3	+2	+1	0	+1	+2	+3	+4
1 Rectal temp (◦C)	*>41*	39–40.9		38–38.9	36–38.4	34–35.9	32–33.9	30–31.9	*<29.9*
2 Mean arterial pressure (mmHg)	*>160*	130–159	110–129		70–109		50–69		*<49*
3 Heart rate (bpm)	*>180*	140–179	110–139		70–109		55–69	40–54	*<39*
4 Respiratory rate (bpm)	*>50*	35–49		25–34	12–24	10–11	6–9		*<5*
5 Oxygen delivery (mL/min)	*>500*	350–499	200–349		*<200*				
6 PO2 (mmHg)					*>70*	61–70		55–60	*<55*
7 Arterial pH	*>7.7*	7.6–7.69		7.5–7.59	7.3–7.49		7.25–7.3	7.15–7.2	*<7.15*
8 Serum sodium (mmol/L)	*>180*	160–179	155–159	150–154	130–149		120–129	111–119	*<110*
9 Serum potassium (mmol/L)	*>7*	6–6.9		5.5–5.9	3.5–5.4	3–3.4	2.5–2.9		*<2.5*
10 Serum creatinine (mg/dL)	*>3.5*	2–3.4	1.5–1.9		0.6–1.4		*<0.6*		
11 Hematocrit (%)	*>60*		50–59.9	46–49.9	30–45.9		20–29.9		*<20*
12 White cell count (103/mL)	*>40*		20–39.9	15–19.9	3–14.9		1–2.9		*<1*

			Age Points						
Age									Points
<44									0
45–54									2
55–64									3
65–74									5
>75									6

	Chronic Health Points	
History of Severe Organ Insufficiency		Points
Nonoperative patients		5
Emergency postoperative patients		5
Elective postoperative patients		2

For assessing the predictive accuracy of individual parameters the following cut-off values were fixed.

1. Packed cell volume (PCV) >44(As suggested by Atlanta symposium)
2. Total WBC count (TC) > 15000 cells/cumm(Imries criteria)
3. Age >55 (Imries criteria)
4. Random blood sugar (RBS) > 200mg/dl(Imries criteria)
5. Blood urea >45mg/dl(Imries criteria)
6. Serum creatinine >2 mg/dl
7. Serum bilirubin > 2mg/dl
8. Alaline aminotransferase (ALT) >200U/L (Imries criteria)
9. Aspartate Aminotransferase (AST) >200 U/L (Imries criteria)
10. Serum calcium (S.Ca++) <8 mg/dl(Imries criteria)
11. Serum Lactate Dehydrogenase (S.LDH) >350units/L (Ransons Criteria)
12. Pa O2<60 mm Hg(Imries criteria)

All these patients were followed up until death / discharge.

Severe acute pancreatitis is defined by the presence of organ failure (shock, pulmonary insufficiency and renal failure) and/or local complications especially pancreatic necrosis but also psuedocyst or abscess or ICU stay of more than 7 days.

12 individual parameters which are used in these scoring systems were assessed for their accuracy in predicting mortality / severity by calculating ‘p’ values. Base deficit and Fluid Sequestration was not assessed for their predictive accuracy as they were used only in a single scoring system ie Ransons scoring system

The sensitivity, specificity, positive predictive value and negative predictive values were calculated using the following formulas.

$$\text{Sensitivity} = \frac{\text{True positive}}{\text{True positive + False Negative}}$$

$$\text{Specificity} = \frac{\text{True Negative}}{\text{True Negative + False Positive}}$$

$$\text{Positive predictive value} = \frac{\text{True Positive}}{\text{True Positive + False Positive}}$$

$$\text{Negative predictive value} = \frac{\text{True negative}}{\text{True Negative + False Negative}}$$

Statistical Tools

The information collected regarding all the selected cases were recorded in a Master Chart. Data analysis was done with the help of computer using **Epidemiological Information Package (EPI 2010)** developed by Centre for Disease Control, Atlanta.

Using this software range, frequencies, percentages, means, standard deviations, chi square and 'p' values were calculated by One way ANOVA and ‘t’ test. Kruskul Wallis chi-square test was used to test the significance of difference between quantitative variables and Yate’s chi square test for qualitative variables.

A 'p' value less than 0.05 is taken to denote significant relationship.

RESULTS

TABLE – 1

AGE VS SEVERITY

Age	Death	SAP	MILD
< 55 (35)	4	5	26
> 55 (15)	4	5	6

Death + SAP Vs Survival 'p' value = 0.046 - Significant

Age > 55 years is significantly associated with an increased mortality & severity

TABLE – 2

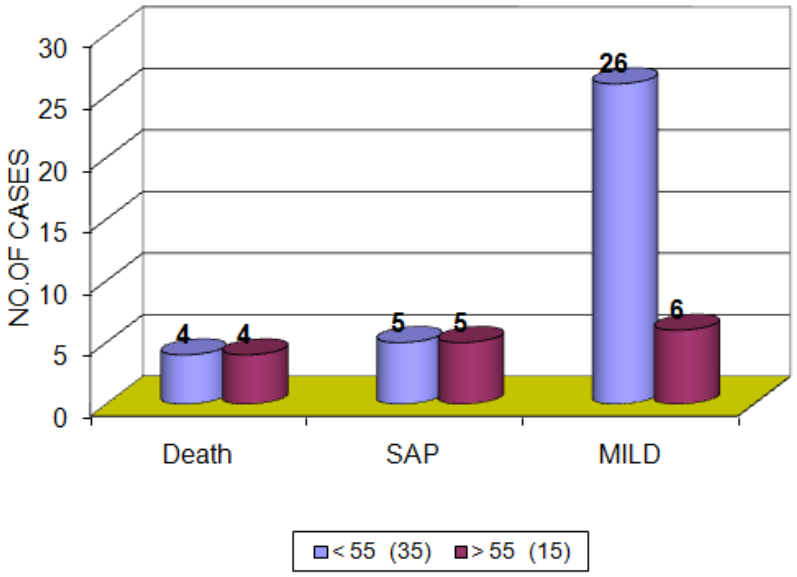
TC VS SEVERITY

Tc	Death	SAP	MILD
< 15000 (41)	2	7	32
> 15000 (9)	6	3	0

Death + SAP Vs Survival 'p' value = < 0.001 - Significant

Leukocytosis > 15000cells/cmm is found to have statistically significant association with mortality and severity.

AGE VS SEVERITY



TC VS SEVERITY

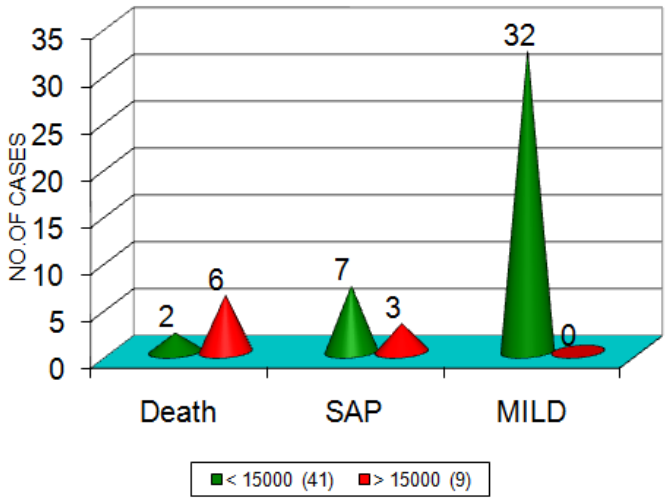


TABLE – 3

RBS VS SEVERITY

RBS	Death	SAP	MILD
< 180(41)	5	7	29
> 180 (9)	3	3	3

Death + SAP Vs Survival 'p' value = 0.083 - Not Significant

With a cut off value of 180 mg/dl the difference in the RBS levels between two groups (Death+SAP Vs Mild) were not statistically significant.

TABLE – 4

ALBUMIN VS SEVERITY

ALBUMIN	Death	SAP	MILD
< 3.2 (9)	3	2	4
> 3.2 (41)	5	8	28

Death + SAP Vs Survival 'p' value = 0.334 - Not Significant

With a cut off value of 3.2 mg/dl the difference in the Albumin levels between two groups (Death+SAP Vs Mild) were not statistically significant.

TABLE – 5

SERUM CALCIUM VS SEVERITY

Sr. Cal	Death	SAP	MILD
< 8 (13)	5	3	5
> 8 (37)	3	7	27

Death + SAP Vs Survival 'p' value = 0.048 - Significant

Serum Calcium>8 mg/dl is found to have statistically significant association with mortality and severity.

TABLE – 6

AST VS SEVERITY

AST	Death	SAP	MILD
< 200 (42)	5	9	28
> 200 (8)	3	1	4

Death + SAP Vs Survival 'p' value = 0.618 - Not Significant

With a cut off value of 200U/Lthe difference in the AST levels between two groups (Death+SAP Vs Mild) were not statistically significant.

SERUM CALCIUM VS SEVERITY

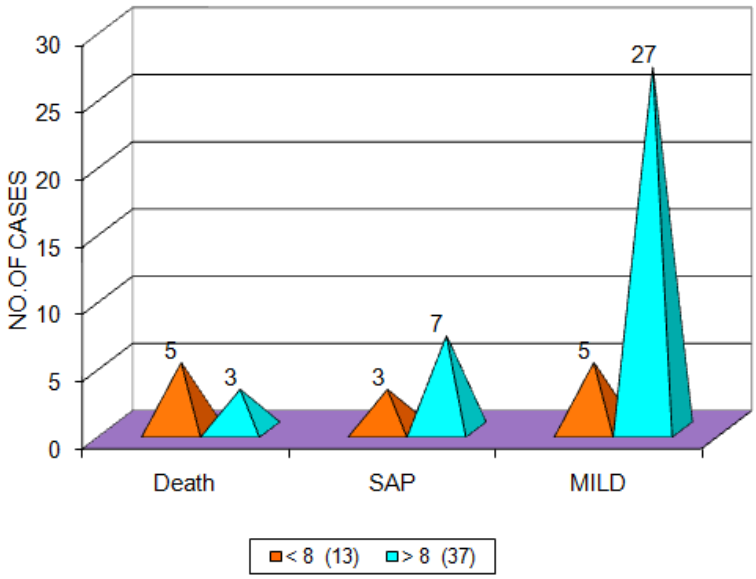


TABLE – 7

ALT VS SEVERITY

ALT	Death	SAP	MILD
< 200 (42)	5	9	28
> 200 (8)	3	1	4

Death + SAP Vs Survival 'p' value = 0.618 - Not Significant

With a cut off value of 200U/L the difference in the ALT levels between two groups (Death+SAP Vs Mild) were not statistically significant.

TABLE – 8

UREA VS SEVERITY

UREA	Death	SAP	MILD
< 45 (27)	4	2	21
> 45 (23)	4	8	11

Death + SAP Vs Survival 'p' value = 0.047 - Significant

Blood Urea > 40mg/dl is significantly associated with an increased mortality & severity

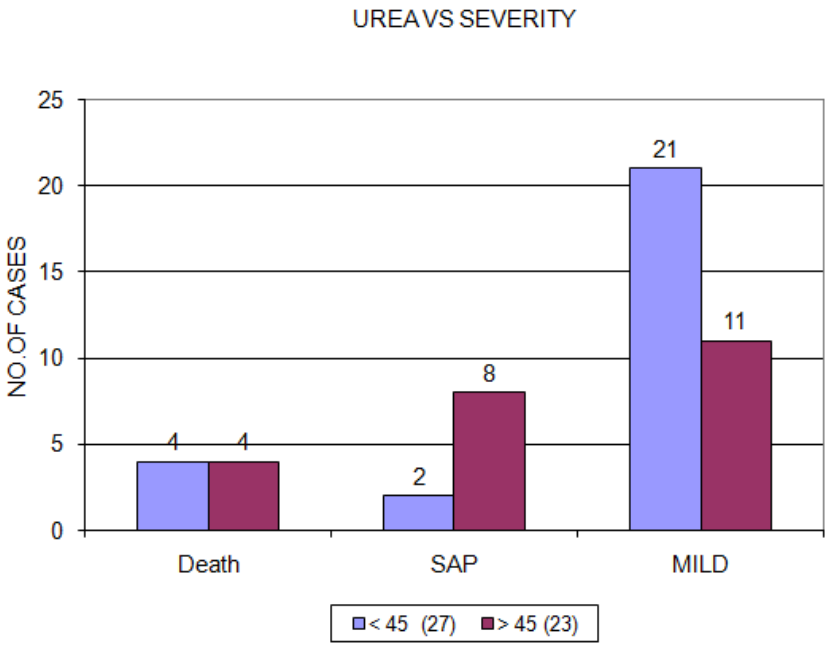

UREA VS SEVERITY
NO.OF CASES
25
20
15
10
5
0
4
4
2
8
21
11
Death
SAP
MILD
< 45 (27)
> 45 (23)

TABLE – 9

CREATININE VS SEVERITY

Creatinine	Death	SAP	MILD
< 2.0 (38)	5	5	28
> 2.0 (12)	3	5	4

Death + SAP Vs Survival 'p' value = 0.028 - Significant

Serum Creatinine >2.0 mg/dl is found to have statistically significant association with mortality and severity.

TABLE – 10

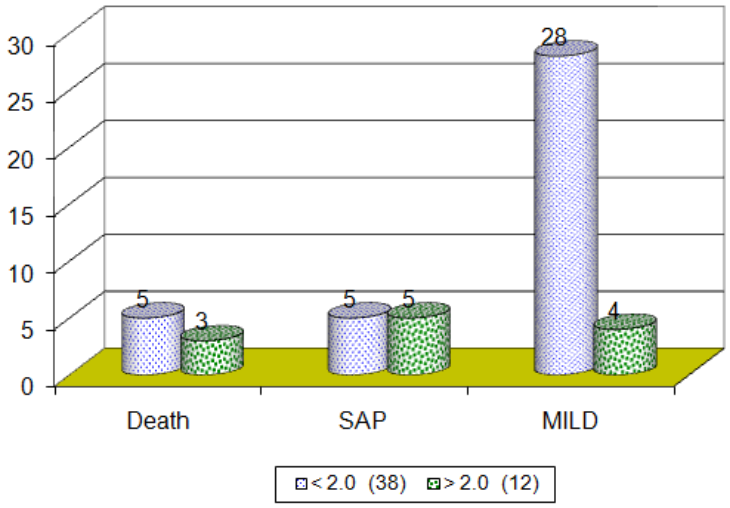
PaO2 VS SEVERITY

PaO2	Death	SAP	MILD
< 60 (7)	7	0	0
> 60 (43)	1	10	32

Death + SAP Vs Survival 'p' value = < 0.001 - Significant

Hypoxia marked by PaO2< 60mm Hg is significantly associated with an increased mortality & severity

CREATININE VS SEVERITY



PAO2 VS SEVERITY

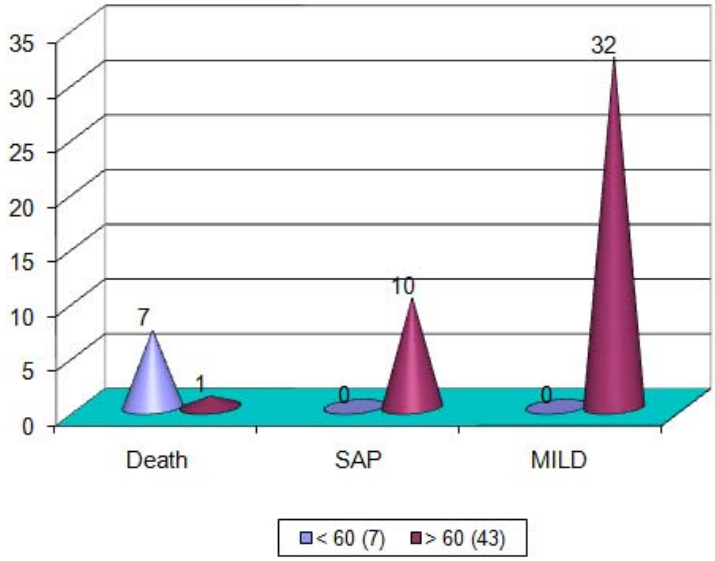


TABLE – 11

LDH VS SEVERITY

LDH	Death	SAP	MILD
< 350 (39)	3	7	29
> 350 (11)	5	3	3

Death + SAP Vs Survival 'p' value = 0.012 - Significant

Serum LDH> 350U/L is found to have statistically significant association with mortality and severity.

TABLE – 1 2

BILIRUBIN VS SEVERITY

Bilirubin	Death	SAP	MILD
< 2 (43)	5	10	28
> 2 (7)	3	0	4

Death + SAP Vs Survival 'p' value = 0.986 - Not Significant

With a cut off value of 2mg/dl the difference in the bilirubin levels between two groups (Death+SAP Vs Mild) were not statistically significant.

LDH VS SEVERITY

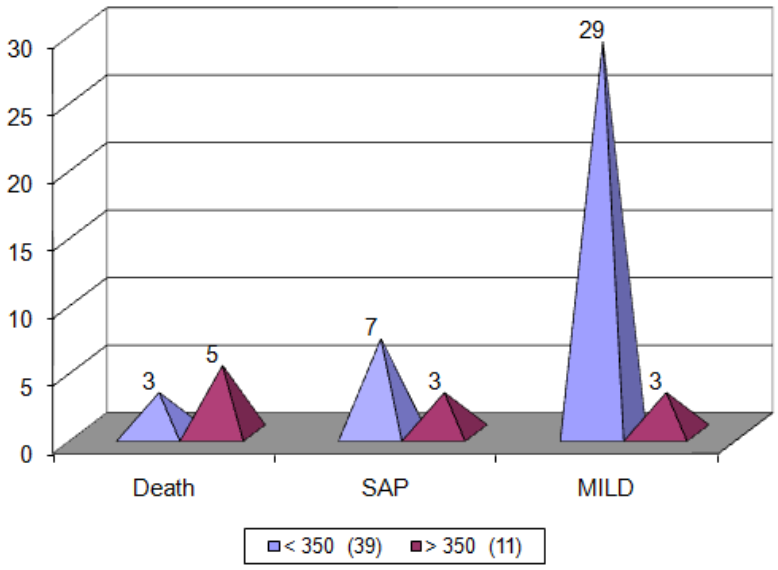


Table :13

RANSON'S SCORE

	Total N = 50	Death : 8	> 7 days / SAP	< 7 days
≥3	16	8	7	1
< 3	34	0	3	31

A total of 16 patients had Ranson's score ≥ 3 which included 15 patients of severe pancreatitis(including death) & 1 patient with mild pancreatitis.

Table :14

	Mortality	Severity
Sensitivity	8 / 8+0×100 = 100	15/15+3×100 = 83.33
Specificity	34/34+8×100 = 80.95	31 / 31+1×100 = 96.8
PPV	8 / 8+8 ×100 = 50	15/15+1×100 = 93.75
NPV	34 / 34+0 ×100 = 100	31 / 31+3×100 =91.8

Ranson's Score has 100% sensitivity and 80.95% specificity in predicting Mortality.Sensitiviy and Specicificity in Predicting Severity is 83.33% and 96.8% respectively

Table :15

APACHE II

	Total N = 50	Death : 8	> 7 days / SAP	< 7 days
>8	15	8	6	1
< 8	35	0	4	31

A total of 15 patients had APACHE II score >8 which included 14 patients of severe pancreatitis(including death) & 1 patient with mild pancreatitis.

Table :16

	Mortality	Severity
Sensitivity	8 / 8+0×100 = 100	14/14+4×100 = 77.78
Specificity	35/35+7×100 = 83.33	31 / 31+1×100 = 96.88
PPV	8 / 8+7×100 = 53.33	14/14+1×100 = 93.33
NPV	35 / 35+0×100 = 100	31 / 31+4×100 =88.57

APACHE II Score has 100% sensitivity and 83.33% specificity in predicting Mortality.Sensitiviy and Specicificity in Predicting Severity is 77.78% and 96.8% respectively.

Table 17

Imries

	Total N = 50	Death : 8	> 7 days / SAP	< 7 days
≥ 3	10	6	4	0
< 3	40	2	6	32

A total of 10 patients had Imrie's score ≥ 3 which and all of them had severe pancreatitis. No patient with mild pancreatitis had a score≥3

Table : 18

	Mortality	Severity
Sensitivity	6 / 6+2 ×100 = 75	10/10+8×100 = 55
Specificity	38/38+4×100 = 90.48	32 / 32+0×100 = 100
PPV	6 / 6+4 ×100 = 60	10/10+0×100 =100
NPV	38 / 38+2 ×100 = 95	32 / 32+8×100 = 80

Imrie's Score has 75% sensitivity and 90.48% specificity in predicting Mortality.Sensitiviy and Specicificity in Predicting Severity is 55% and 100% respectively

Table :19

Bank's Scores

	Total N = 50	Death : 8	> 7 days / SAP	< 7 days
≥ 1	25	8	6	11
< 1	25	0	4	21

A total of 25 patients had Bank's score ≥ 1which included 14 patients of severe pancreatitis(including death) & 11 patient with mild pancreatitis.

Table:20

	Mortality	Severity
Sensitivity	8 / 8+0×100 = 100	14/14+4×100 = 77.78
Specificity	25/ 25+17×100 = 59.52	21 / 21+11×100 = 65.63
PPV	8 / 8+17×100 = 32	14/14+11×100 =56
NPV	25 / 25+0×100 = 100	21 / 21+4×100 = 84

Bank's Score has 100% sensitivity and 59.52% specificity in predicting Mortality.Sensitiviy and Specicificity in Predicting Severity is 77.78% and 65.63% respectively

Table:21

Pitchumani & Agarwal Score

	Total N = 50	Death : 8	> 7 days / SAP	< 7 days
≥ 1	20	8	3	9
< 1	30	0	7	23

A total of 20 patients had Pitchumani score ≥ 1which included 11 patients of severe pancreatitis(including death) & 9 patient with mild pancreatitis.

Table :22

	Mortality	Severity
Sensitivity	8 / 8+0×100 = 100	11/11+7 ×100 = 61.11
Specificity	30/30+12×100 = 71.43	23 / 23+9×100 = 71.88
PPV	8 / 8+2×100 = 40	11/11+9×100 =55
NPV	30 / 30+0×100 = 100	23 / 23+7×100 = 76.67

Pitchumani and Agarwal Score has 100% sensitivity and 71.43% specificity in predicting Mortality.Sensitiviy and Specicificity in Predicting Severity is 61.11% and 71.88% respectively.

Table:23

Comparison of Scoring systems in predicting Mortality

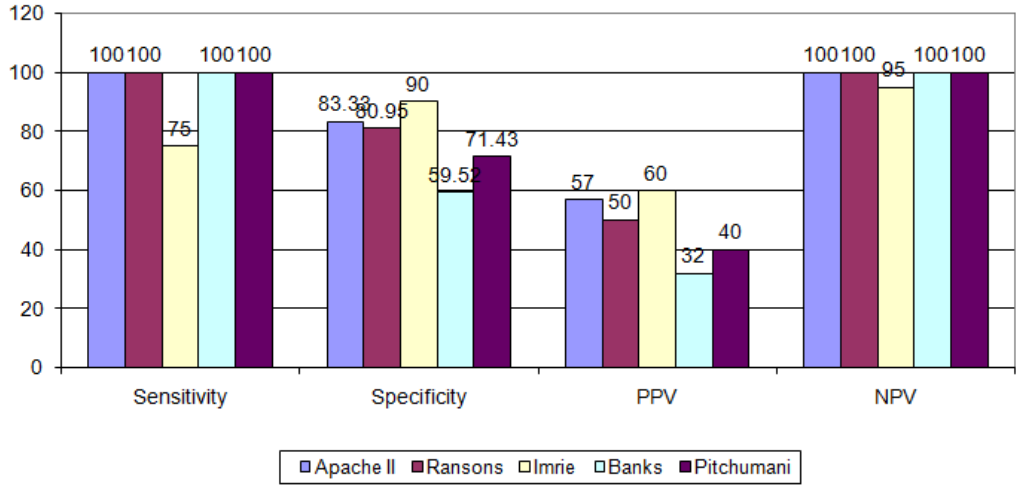
	Sensitivity	Specificity	PPV	NPV
Apache II	100%	83.33%	57.33	100
Ransons	100%	80.95%	50	100
Imrie	75%	90%	60	95
Banks	100%	59.52%	32	100
Pitchumani	100%	71.43%	40	100

Table:24

Comparison of Scoring systems in predicting Severity

	Sensitivity	Specificity	PPV	NPV
Apache II	77.78%	96.88%	93.33	88.57
Ransons	83.33%	96.88%	93.75	91.18
Imrie	55%	100%	100	80
Banks	77.78%	65.63%	56	84
Pitchumani	61.11%	71.88%	55	76.67

MORTALITY



SEVERITY

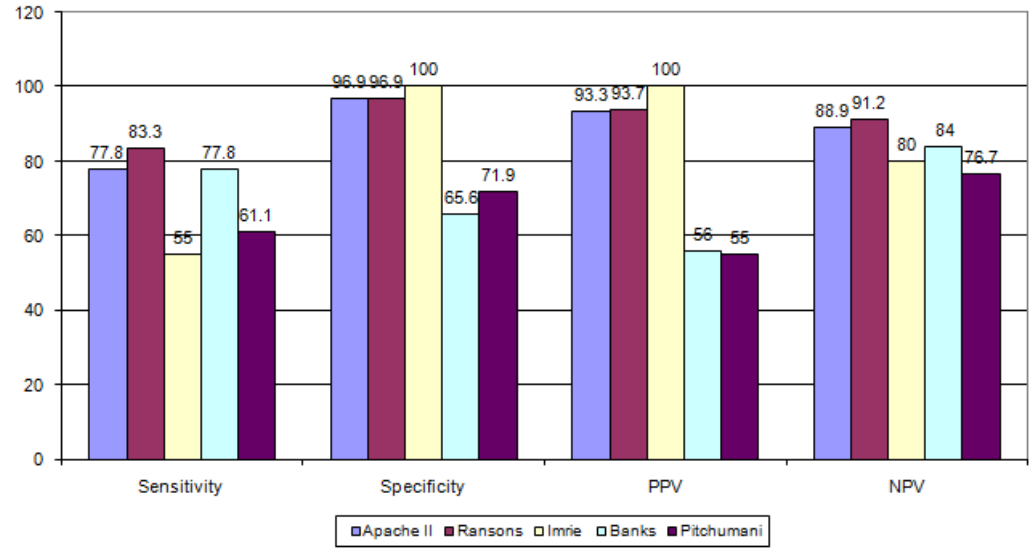


Table :25

Comparison of performance APACHE II system in various studies in predicting severity

	Our study	Papachriston Et al[10]	Wilson C Et al [32]	Larvin et al[41]	Marco Simoe[51]
Sensitivity	77.78%	70.3%	82%	77%	79.4%
Specificity	96.88%	71.9%	74%		83.1%

Table :26

Comparison of performance of Ranson's system in various studies in predicting severity

	Our Study	Papachriston Et al[10]	Sternberg[44]	Marco Simoe[51]
Sensitivity	83.33%	84.2%	72%	91.2%
Specificity	96.8%	89.8%	76%	71.4%

Table :27

Comparison of Performance of Imrie's system in various studies in predicting severity

	Our Study	Barreto Et al[6]	Sternberg[44]	Marco Simoe[51]
Sensitivity	55%	56%	63%	73.5%
Specificity	100%	98%	84%	71 %

DISCUSSIONS

In this study we compared the accuracy of five representative prognostic multi factorial scoring systems in acute pancreatitis. We also assessed the accuracy of 12 individual parameters in assessing the severity / mortality of acute pancreatitis.

50 patients with acute pancreatitis were enrolled in our study. Among this 42 were males and 8 were females.

In this 50 patients, 8 patients died ie the mortality rate was 16%. A total of 18 patients (including dead patients) were found to have severe acute pancreatitis (SAP).

On assessing the individual variable, we found that there was statistically significant difference exist between severe pancreatitis and mild pancreatitis in the case of seven variables.

They include

1. PaO2 - 'p' value < 0.001
2. Blood Urea - 'p' value 0.047
3. Sr.creatinine- 'p' value 0.028
4. Sr. Calcium- 'p' value 0.048

5. Total count - 'p' value < 0.001

6. LDH - 'p' value 0.012

7. Age - 'p' value 0.046

In a study conducted by Blamy et al[19], they found that Sr.Creatinine of > 2.0 mg/dl is associated with severity. Fan[31] and coworkers also suggested that isolated serum urea elevation could predict SAP. Similiar finding were alsoobserved in studies by Lautz et al[38], Haxiaobieke Kasimu et al[15], Sournitra R Ecachempali[3] et al.

In our study hypoxia ie PaO2 <60mm Hg is also found out be a strong predictor of SAP.Similiar observations were made by Sournitra Ecachempali[3] et al.

Hypocalcemia also was found to be a predictor of severity of pancreatitis in our study.This is correlating with the various studies by Bechien.WU et al[57], Cooper MJ et al[55], Sournitra R Ecachempali[3] et al.

Serum LDH levels also showed a significant difference between mild and severe acute pancreatitis.Similiar observations were made by Kaya E et al[49], Chen C-C et al[45].

Leukocytosis which is considered as a marker of SIRS is also an independent predictor of SAP.With a cut off value of >15000 it also showed a significant difference between Mild and Severe

pancreatitis.Similiar findings were observed by Lautz et al[38], Haxiaobieke Kasimu et al[15].

Hemoconcentration marked as an increase in PCV is generally considered as a marker of severity. In our study none of the patients had PCV more than 44.This is not correlating with the various literature.

On analyzing various scoring system, a Ranson's score ≥ 3, Apache II > 8, Imrie score ≥ 3, Banks score ≥ 1, Pitchumani & Agarwal score ≥ 1 were taken as predictor of severe pancreatitis.

Apache II Score :

Among 50 patients, 15 patients had an Apache II score of >8. It included all the dead patients ,that means Apache II had 100% sensitivity in predicting mortality with a negative predictive value of 100. Its specificity was 83.33% of positive predictive value was 53.33%.

This results corresponds to the results of Papachisto et[10] al were they found that Apache II has a 100% sensitivity in predicting mortality.

A total of 18 patients had severe acute pancreatitis (including death patients). In this 14 patients had Apache II > 8, among

patients with mild pancreatitis only one had Apache II score of >8. Thus on predicting severity Apache II had a sensitivity of 77.78%, specificity of 96.88%, PPV of 93.33 and NPV of 88.57.

Wilson C et al[32], in their study found that Apache II has a sensitivity of 82%, specificity of 74%.

Study by Larvin et al[41] also showed that Apache II has a sensitivity of 77% at the time of admission.

Papachriston et[10] al in their study also showed that Apache II has a sensitivity of 70.3% and specificity of 71.9%.

Marco Simoe et al[51] in their study showed that APACHE II system has

Sensitivity of 79.4% and specificity of 83.1% in predicting severity.

In our study, the sensitivity very much matches with these studies, and the specificity in our study was more than what we found in many other studies.

Ranson's Score :

Among 50 patients, 16 had a Ranson's score ≥ 3, Among them 8 patients died 7 had other evidences of SAP and 1 had mild pancreatitis that means all the dead patients had a Ranson's score of

≥ 3. ie. It has a 100% sensitivity in predicting mortality as we have seen with Apache II. The specificity was 80.95%.

In predicting the severity as a whole its sensitivity was 83.33% specificity of 96.8% PPV of 93.15 and NPV of 91.18.

In similar study, Georgios L. Papachriston et[10] al showed that the sensitivity of Apache II in predicting the mortality is 100% as we have seen in our study. While the sensitivity in predicting the severity was 84.2% and specificity was 89.8% which is very much similar to our study.

In a study by Steinberg[44] Ranson criteria have an estimated sensitivity of 72% and specificity of 76%.

Marco Simoe et al[51] in their study showed that Ransons system has a sensitivity of 91.2% and specificity of 71.4% in predicting severity.

A meta analysis encompassing 1,300 patents reported that Ranson's has an overall sensitivity of 75% and specificity of 77%.

Imrie's Scoring system :

Using a cut off of ≥ 3 as severe pancreatitis, Imries scoring system predicted severe pancreatitis in 10 out of 50 patients.

Death patients - n = 8 - among this 6 had Imrie score ≥ 3

SAP other than Death- n = 10 – Among this 4 had Imrie score ≥ 3

Mild pancreatitis - n = 32 - Among this none had score ≥3

With this data we found that Imries system has a sensitivity of 75, specificity of 90.48%, PPV of 66 and NPV of 95 in predicting mortality.

For assessing the severity it has a sensitivity of 55%, specificity of 100%, PPV of 100 and NPV of 80. Our study is very much comparable to the study Berreto et al[6] done in Goa Medical College, India were they found that Imries system has a sensitivity of 56%, specificity of 98%, PPV 94% and NPV of 80%.

Marco Simoe et al[51] in their study showed that Imrie's system hassensitivity of 73.5% and specificity of 71.1% in predicting severity.

In a study by Steinberg[44] Imrie'scriteria have an estimated sensitivityof 63% and specificity of 84%.

But when the scoring system was originally proposed by SL Blamey, CW. Imrie in 1986, it predicted severity correctly in 79% cases[19].

Bank's Scoring system :

A bank's score ≥ 1 was taken as a predictor of severe acute pancreatitis. With this cut off it predicted SAP in 25 out of 50 patients.

Death – 8 patients – all of them had Bank's score ≥1

Severe Acute pancreatitis (excluding death) → 10 patients

6 of them had score ≥1

The sensitivity in predicting the mortality was 100% while the specificity was only 59.52%. On predicting severity sensitivity was 77.77%, specificity was 65.63%.

Pitchumani and Agarwal Score :

A score ≥1 was taken as predictor of severe acute pancreatitis with this 20 out of 50 patients were predicted to have SAP.

Death - n = 8 All of them had Score ≥1

SAP - n = 10 - 3 had Score ≥ 1

Mild - n = 32 - 9 had score ≥1

In predicting mortality sensitivity was 100%, specificity was 71.45%.

In predicting severity it had a sensitivity of 61.11%, specificity of 71.88%.

On comparing the different scoring systems, we found that in predicting mortality, Apache II, Ranson's, Bank's, Pitchumani scoring systems had 100% sensitivity while the maximum specificity was for Imries.

But for predicting the severity compared Apache II, Ranson's system, performed well with a sensitivity of 83.33% and specificity of 96%. Imries system was highly specific but its sensitivity was quite low. Both Bank's system and Pitchumani and Agarwal scoring system has relatively low sensitivity and specificity.

The APACHE II System seems to be superior to other systems because it is the only system which takes in to account of all the major risk factors that predict outcome from the disease including the acute physiological changes as well as the patient's ability to recover which may be diminished by advancing age and chronic diseases.

Our study also showed that still now Ranson's score remains valid for predicting the severity and mortality of acute pancreatitis. It was proved to be equally efficient when compared to the rather complex APACHE II system in predicting SAP.

CONCLUSION

1. Evidence of end organ dysfunction marked by hypoxia and acute renal failure are highly sensitive predictors of severity and mortality of acute pancreatitis.
2. Advancing age, leukocytosis, hypocalcemia and increase in LDH levels were the other factors found to be significant in predicting severity of acute pancreatitis in our study.
3. Ranson's scoring system was found to be the best to predict the outcome in acute pancreatitis compared to Apache II in our study.
4. Imrie scoring system even though highly specific it is less sensitive in predicting outcome in our study.
5. Bank's system and Pitchumani and Agarwal scoring system had low sensitivity and specificity.
6. The limitations of these scoring systems could be that they converted continuous variables to binary variables of equal weight and thus failing to capture synergistic effects based on the interaction of inter dependent systems.

7. Future researches could focus on the incorporation of pre-existing risk factors and novel accurate biomarkers into the scoring systems.

LIMITATIONS OF THE STUDY

1. Sample size in our study is relatively small.
2. The aetiology of pancreatitis was not considered in our study.

SUMMARY

A prospective, observational study was conducted in 50 patients admitted with acute pancreatitis in Government Rajaji Hospital Madurai. The aim of the study was to compare the strength of various parameters in predicting the severity of acute pancreatitis and to compare the performance of various scoring systems predicting the mortality and severity of acute pancreatitis. On analyzing the final results we found that Ranson's scoring system and Apache II system were the best systems in predicting the mortality and severity of acute pancreatitis. Among individual parameters hypoxia, renal failure, advancing age, leukocytosis, hypocalcemia and increase in LDH levels were associated with increased severity of acute pancreatitis.

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A Study on the profile of various scoring systems in assessing the severity of acute pancreatitis

Name: Age: Sex:

Presenting complaints: Alcoholic:

GCS	Pulse Rate	B.P	Respiratory Rate	Temperature	Comorbidity

Investigations

PCV		S.Amylase		S.Bilirubin		USG Abdomen
TC		S.Calcium		AST		
PLC		S.Albumin		ALT		CT Abdomen
PaO2		Blood Urea		LDH		
HCO3-		S.Creatinine		RBS		Fluid sequestration

Scores

APACHE II	Ranson's	Imries	Banks	Pitchumani

OUTCOME

S.No.	Name	Age	sex	BP	RR	GCS	PCV	TC	PLC	PaO2	AMYLASE	RBS	Bilirubin	Calcium	Albumin	AST	ALT	LDH	Urea	S.No.
1	Shanmuganathan	65	M	90/70	32	12	30	17400	2.1	54%	612	106	2.9	6.6	4.5	59	49	373	18	1
2	Mahalingam	47	M	100/80	36	13	31	15300	99000	52%	456	460	0.9	8.1	4.2	21	53	750	132	2
3	Sivakumar	56	M	90/60	26	15	36	15500	1.3l	59.50%	1116	480	1	7.9	4.8	62	98	850	103	3
4	Baskaran	32	M	110/70	28	15	30	17900	1.6L	58%	846	108	2.2	8.2	3.9	286	302	410	86	4
5	Ramdoss	48	M	120/80	34	15	24	19070	1.2l	52	555	48	0.9	7.1	3	34	22	420	17	5
6	Periyakaruppan	35	M	96/60	32	14	40	13300	100000	50	566	112	0.8	7.8	3.1	320	344	365	30	6
7	Bhoomi	60	M	80/70	22	14	33	12500	78000	65%	330	84	2.1	7.8	3.1	376	360	280	24	7
8	Thavasi	62	M	110/70	30	15	34	18200	2.2L	56%	724	356	1.3	8.4	4.2	120	98	165	146	8
9	Muthukon	58	M	100/60	20	15	30	11200	1.1l	68%	448	120	0.8	7.5	3	56	78	180	88	9
10	Vijayan	56	M	130/80	20	15	31	11200	2.4L	72%	656	112	1	7.4	4.3	88	38	408	102	10
11	Palani	62	M	126/70	26	15	32	15660	88000	70%	342	256	1.8	8.4	3.8	224	312	112	112	11
12	Ayyanar	35	M	116/80	16	15	41	4500	3.2L	56%	512	98	1	7.1	5.5	65	80	240	123	12
13	Arasan	58	M	90/60	18	15	36	15420	4L	68%	646	208	0.9	8.2	4.8	78	45	389	56	13
14	Muthusamy	50	M	100/60	18	15	31	6330	1.1L	70%	387	121	0.8	8.9	5	86	64	210	50	14
15	Ramakrishnan	56	M	110/80	22	15	30	7200	1.6L	65%	442	229	1	8	4.8	42	28	412	112	15
16	Pavunraj	60	M	96/60	24	15	38	16680	90100	70%	586	98	1.1	7.3	3.1	112	89	121	24	16
17	Ramayee	48	F	100/80	22	15	62	6800	4.1L	72%	360	105	1	8	4.2	45	23	408	30	17
18	Mani	50	M	120/80	16	15	32	9600	2.5L	68%	286	110	1.1	8.4	4.5	32	28	128	48	18
19	Abdulla	35	M	130/80	14	15	38	4200	3.5L	74%	312	154	0.8	7.8	4.2	21	18	98	16	19
20	Arumugam	36	M	140/80	12	15	38	5600	3.9L	74%	778	80	0.8	7.1	4.6	27	28	110	84	20
21	Maniraj	46	M	120/70	16	15	32	9200	2.8L	68%	230	84	0.9	7.5	5.4	42	62	140	25	21
22	Saravanamuthu	43	M	118/60	12	15	52	5800	2.1L	70%	263	110	0.8	8.4	4.5	336	426	96	34	22
23	Annadurai	38	M	138/90	14	15	33	7200	2.6L	64%	446	96	1	8.1	4	46	56	156	56	23
24	Sivalakshmi	46	F	112/79	15	15	36	5800	1.9L	68%	447	68	0.8	8.2	5.8	17	20	53	17	24
25	Palraj	22	M	120/80	13	15	34	9000	4.3L	71%	462	123	1.2	8.1	3	21	46	146	21	25
26	Subramaniam	56	M	120/70	11	15	30	12000	1.8L	72%	322	140	0.8	8.8	4	229	229	110	48	26
27	Matchakalai	35	M	118/90	10	15	32	7200	2.0L	70%	1404	86	2.2	8	4.5	196	145	127	25	27
28	Duraipandi	35	M	112/70	18	15	36	6200	1.9L	72%	767	102	2.8	8.6	4.5	82	65	180	24	28
29	Sundaresan	30	M	110/70	16	15	33	5200	3.4L	66%	289	79	1.2	8	3.8	21	25	108	32	29

30	Bhuvaneshwari	42	F	140/90	17	15	42	4600	2.2l	70%	382	110	1	8.2	4	42	36	428	28	30
31	Palanivel	29	M	150/90	14	15	44	4100	2.6l	72%	347	146	3	7	3.1	45	21	386	23	31
32	Muthukrishnan	52	M	120/80	13	15	30	6000	3.4l	68%	426	125	1	8.4	4.5	62	48	189	46	32
33	Rahman	36	M	118/60	12	15	36	4800	4l	64%	560	90	0.8	8.4	5.2	36	29	140	56	33
34	Ramalakhmi	48	F	120/80	16	15	32	5110	1.9L	72%	496	146	0.8	8.1	3.9	46	36	196	96	34
35	Sundar	42	M	150/100	17	15	33	6700	3.4L	70%	296	161	1	9	5.5	65	46	234	116	35
36	Raja	34	M	130/80	12	15	40	6200	2.8L	72%	280	154	1	9	4.5	41	30	124	26	36
37	Palaniyandi	38	M	120/80	20	15	34	4680	3.3L	67%	223	93	0.8	8	3.5	320	321	90	18	37
38	Panchavarnam	56	F	120/60	14	15	30	8200	1.8L	70%	679	202	1	8.1	4.5	46	64	126	39	38
39	Thevanesam	42	M	120/60	17	15	33	10200	1.6L	67%	356	110	2.6	8.2	3.9	286	268	110	26	39
40	Rajaram	37	M	110/70	13	15	36	11000	4.1L	70%	421	90	1	7.6	3	45	28	100	48	40
41	Karupusamy	32	M	130/90	21	15	40	5200	2.2L	64%	650	225	1	8	3.8	36	46	390	28	41
42	Selvi	60	F	130/100	12	15	33	4790	2.9L	68%	268	89	1.1	8.2	4.8	56	26	120	48	42
43	Jeyalakshmi	63	F	110/80	14	15	36	5600	4.1L	72%	330	120	1	8.1	5.5	84	76	150	86	43
44	Anthoniammal	42	M	110/90	17	15	33	5270	3.3L	68%	268	110	0.9	8	5.4	25	16	90	34	44
45	karuthagoundar	28	M	140/90	18	15	40	4600	3.1L	72%	332	96	1	8.6	4.4	46	46	108	30	45
46	Andy	39	M	140/90	9	15	36	4870	2.1L	68%	423	78	1.1	8	4.5	68	87	119	46	46
47	Pandian	36	M	112/80	12	15	33	5280	1.8L	70%	225	90	1	8.1	5	56	36	157	35	47
48	Kalpana	57	F	120/80	10	15	40	7400	2.1L	66%	345	108	0.9	8.4	3.1	76	56	118	26	48
49	Sureshkumar	61	M	110/90	16	15	38	6580	2.8L	68%	280	208	1.1	8	4.6	66	25	256	38	49
50	Kumar	36	M	118/60	12	15	36	4800	4l	64%	560	90	0.8	8.4	5.2	36	29	140	40	50

Creatinine	Base Deficit	Fluid Seques	.Sodium	potassium	Survival	Ransons	Imries	Banks	Pitchumani	Apache
0.9	3	4L	134	3.3	DEATH	4	4	2	2	12
3.8	2	3.5L	126	3.5	DEATH	4	4	3	2	18
2.8	3	4L	135	4	DEATH	5	5	3	2	15
1.4	3	3.5L	145	4.4	DEATH	3	2	2	2	15
0.5	2	3.5L	130	4.2	DEATH	3	3	2	2	15
0.9	2	4L	127	4.1	DEATH	3	3	2	2	14
0.8	3	5L	138	3.7	DEATH	4	3	2	2	14
2.6	2	3L	125	5.4	DEATH	3	2	2	1	15
2	1	3L	120	5.3	>7DAYS	3	3	2	2	8
2.5	0	3.5L	138	4.2	>7DAYS	3	3	2	1	8
3.8	0	3L	142	5.2	>7DAYS	3	3	1	0	8
3.6	1	3L	132	4.8	>7DAYS	3	3	1	0	8
1.1	1	3L	130	4.3	>7DAYS	3	2	1	0	8
0.9	0	3.5L	133	3.6	>7DAYS	0	0	0	0	4
2.8	1	3.5L	142	5.8	>7DAYS	3	2	0	0	8
1	0	3L	128	3.6	>7DAYS	3	2	1	1	5
1.1	0	4L	134	3.1	>7DAYS	1	0	0	0	4
1.2	0	2.5L	129	5.6	>7DAYS	0	0	0	0	5
0.7	0	2L	134	4.6	<7DAYS	1	1	1	1	4
3.1	0	3L	141	4.7	<7DAYS	2	2	2	1	4
0.8	0	2.5L	140	4.1	<7DAYS	1	1	1	1	4
1.1	0	3L	129	5.5	<7DAYS	1	1	0	0	1
1.6	0	3L	139	5.2	<7DAYS	0	0	0	0	3
0.7	0	3L	130	5.1	<7DAYS	0	0	0	0	2
0.8	0	3.5L	133	5	<7DAYS	1	1	1	1	1
1.2	0	2.5L	127	4.8	<7DAYS	0	0	0	0	6
0.9	0	2.5L	143	4.9	<7DAYS	0	0	0	0	0
0.7	0	3L	136	4.8	<7DAYS	0	0	0	0	0
1.2	0	2.5L	132	4.5	<7DAYS	0	0	0	0	1

0.6	0	3L	137	5.1	<7DAYS	0	0	0	0	0
0.8	0	3L	145	4.1	<7DAYS	3	2	1	1	0
1.2	0	3L	128	5.2	<7DAYS	0	0	0	0	3
1	0	3.5L	142	3.7	<7DAYS	0	0	0	0	1
3.4	0	2L	133	5.3	<7DAYS	1	1	1	1	5
3.9	0	3L	143	5.1	<7DAYS	1	1	1	1	8
0.9	0	3L	126	3.9	<7DAYS	0	0	0	0	1
0.7	0	3.5L	137	4	<7DAYS	1	1	0	0	1
1.1	0	3.5L	143	4.2	<7DAYS	1	1	0	0	3
0.6	0	3L	142	4.2	<7DAYS	1	1	0	0	0
1.4	0	3L	127	4	<7DAYS	2	2	2	1	0
0.8	0	2L	136	3.6	<7DAYS	2	1	0	0	0
1.4	0	2.5L	129	3.9	<7DAYS	0	0	0	0	2
2.6	0	2L	131	5.2	<7DAYS	1	1	1	1	6
1.1	0	3L	142	4.8	<7DAYS	0	0	0	0	1
1	0	3.5L	126	4	<7DAYS	0	0	0	0	1
1.4	0	3L	138	5	<7DAYS	0	0	0	0	1
1	0	3L	140	5.1	<7DAYS	0	0	0	0	1
0.9	0	2.5L	139	3.7	<7DAYS	1	1	1	0	2
1.2	0	2.5L	140	3.8	<7DAYS	1	1	0	0	2
1	0	3.5L	142	3.7	<7DAYS	0	0	0	0	1